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Issue Date: 06 October 2006

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In the Matter of

T.S.[1]
Claimant
v.
IMC AGRICO MP, INC./TRAVELERS INSURANCE CO.
Employer/Carrier
And
DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest
……

Case No. 2003 LHC 01686
2005 LHC 02089
OWCP No. 6-187024
6-179845

Decision and Order

This matter arises pursuant to two claims for medical benefits which were consolidated for hearing under the Longshore Act. Claimant is currently receiving permanent total disability compensation for a 1999 spinal injury. Tr. 18. In this proceeding, he is claiming medicals benefits for a pulmonary condition he alleges was caused by chemical exposures in the workplace and a cardiac condition he attributes, in part, to two heart attacks which he alleges were related to his spinal injury and which exacerbated his pre-existing coronary artery disease. Tr. 18-19. The pulmonary claim is designated as 2003 LHC 1686, (OWCP # 06-179845), while the cardiac claim is designated as 2005 LHC 2089, (OWCP # 06-187024).

[1] Beginning on August 1, 2006, administrative law judge decisions rendered in Black Lung Benefits Act and Longshore and Harbor Workers' Compensation Act cases will no longer display the claimant's full name in the decision or in the caption.

The issue is similar in both claims: the etiologies of the pulmonary and cardiac conditions. Tr. 20.

The record shows that Claimant worked over 30 years in jobs involving the storage, manufacturing, and shipping of phosphate products, and loading and unloading ships as a gantry operator. Tr. 12-13. He contends that, as a result of his exposure to chemical dusts and fumes, he developed emphysema, COPD, and occupational asthma. Tr. 13-14. Employer denies that Claimant has occupational asthma and believes his emphysema and chronic obstructive pulmonary disease (COPD) are due a 30-year smoking history which Claimant concedes. Tr. 13; 32-35. Claimant quit smoking in 1997. Tr. 87; Tr.140.

Turning to the cardiac condition, the record shows that Claimant has experienced chronic angina or chest pain since 1997, Tr. 142-43, has a pre-existing coronary condition, and has suffered four heart attacks; one in 1997, one in 2002, and two in 2004. Tr. 141. After the heart attack in 1997,[2] he had a quintuple bypass and placement of stents and was advised to begin a program of exercise. Br at 5; Tr. 14-15. After his back injury in 1999, he was unable to exercise and was prescribed Celebrex for his injuries. Claimant believes his sedentary lifestyle contributed to a worsening of his heart condition and contributed to two heart attacks in August and December, 2004, and that the Celebrex contributed to the coronary blockages that triggered these thrombic events. Tr. 15. In addition, Claimant had to suspend taking his heart medication, Plavix, a blood anticoagulant, in order to undergo injury-related epidural steroid injection therapy in December, 2004. Thereafter, he suffered a heart attack triggered, in part he alleges, by the suspension of Plavix which was a prerequisite to the injury-related epidurals. Tr. 16-18. Claimant argues that since the last heart attack, his condition worsened and he needs more medication. Tr. 199.

Employer responds that Claimant has progressive coronary artery disease, suffered two heart attacks prior to 2004, Tr. 22-23, and it denies that any proof exists that the prescription of Celebrex, the suspension of Plavix, or the sedentary lifestyle caused or contributed to the heart attacks or Claimant's current cardiac condition. Tr. 26-28. Employer believes Claimant's coronary artery disease would

[2] Claimant alleges that the first heart attack occurred at work in 1997, and was work-related, Tr. 199, however, no claim was filed as a result of the coronary incident.

necessitate the same cardiac care he currently receives had he not suffered the heart attacks in 2004. Tr. 29-30; 226.[3]

Findings of Fact

Cardiac Condition

The record shows that Claimant worked for Employer for 32 ½ years. Tr. 92. In December, 1997, he had a heart attack at work. Tr. 93-94. After open heart surgery, following the advice of his doctor, he changed his life and started exercising, and by 1999 was walking four miles per day. Tr. 95-98; 72-3, 86-87. In June 1999, he injured his back at work, and was treated by Dr. Castroni. Tr. 99. Dr. Castroni wanted to perform back surgery, but deferred because he was concerned that Claimant's heart and lungs would not withstand the surgery. Tr. 100. Instead, he placed him on sedentary physical restrictions, Tr. 101; 74, which Claimant caused his overall cardiac condition to deteriorate. Tr. 102.

In March 2002, Claimant believes he may have been on Celebrex prescribed by Dr. Dennison when he had another heart attack. Tr. 102-104. At the time, he was taking Plavix. Tr. 103. In August, 2004 he had gallbladder surgery, and had to suspend the Plavix. Ten days later, he had a heart attack. Tr. 105; 75-76. In December, 2004 Dr. Dennison recommended epidural steroid injections for Claimant's back condition, but was concerned that the Plavix would cause bleeding problems. Tr. 104. As a result, Claimant stopped taking Plavix, aspirin, and vitamin E before the epidurals, Tr. 107, and a few days after the epidurals, the day after Christmas, 2004, he had a heart attack. Tr. 105-106; 76-77.

Claimant testified that he is having more chest pain and takes nitro more often now, fatigues more easily, and is unable to get needed hernia surgery or any more epidurals for his back. Tr. 109-111. Claimant's wife, S.S., testified that he has been depressed since his 2004 heart attacks. Tr. 79.

[3] The parties recently advised that they will be attempting to mediate this matter. Parties are always encouraged to mediate pre-hearing and hopefully resolve their differences. Here, however, the hearing has been held, post hearing briefs have been filed; and a considerable amount of time has been devoted to a careful review of the voluminous evidentiary record the parties developed at the hearing and their lengthy post-hearing briefs. Under these circumstances, further delays are unwarranted. The parties' future efforts to mediate their disputes may be guided by the adjudicative disposition of issues fully litigated in this proceeding.

Pulmonary Condition

Claimant testified that he started working at IMC as a laborer, shoveling wet and dried bulk chemicals including phosphate, DAP (di- ammonium phosphate), GTSP(granular triple super phosphate), AFI products, dyna-phosphate, bi-phosphate, multi-phosphates, a powder ROP (run of the pile) and ROM (run of the mill), phosphate rock, MAP (ammonium phosphate), and other chemicals. Tr. 111-13, 116-22; 149-50. CX 17; *see also,* Tr. 185-86. DAP, MAP, and GTSP are fertilizers. Tr. 189.

The record shows that materials were brought in by truck or railcar and stored in rock or granular form in large warehouses and silos in the yard at the 22 acre marine terminal. Tr. 150-51, 158; Tr. 188. Some were fed into hoppers that moved them to the dry mill to draw moisture out of the chemicals. Tr. 113; 151-52. Once processed through the dry mill, the material was stored in silos and large enclosed warehouses. Tr. 114. Other chemicals, in liquid form, would be piped into storage tanks. Tr. 146. Claimant was exposed to chemical dusts or fumes in virtually all locations he worked at the terminal. 152-54. He shoveled chemicals as a laborer, operated payloaders used to move the material which generated a lot of material dust which was confined within the building or silo in which they were being stored, operated the dry mill and conveyors, and later operated the gantry which loaded the chemicals onto ships for delivery to customers. Tr. 122-23; 153-60; *see also,* Tr. 179-80.

The dry mill was made of fire brick which occasionally would cake with chemicals and which, Claimant, as dry mill operator, occasionally had to break loose with a pick, and, he occasionally, had to replace bricks. Tr. 124-26; Tr. 177. In 1996, the dry mill was shut down. Tr. 129. Claimant then moved to the gantry loading the processed materials onto ships; a process that generated considerable chemical dust . Tr. 127-28. The gantry cab is enclosed. Tr. 144.

Over the years, Employer tried various types of devises and machines to improve air quality, but Claimant testified that nothing worked well until air conditioned equipment cabs were introduced in late 1980's. Tr. 129-30, 144-45; Tr. 148-49; Tr. 178. Although the dust situation improved over time, Tr. 147; *see also* Tr. 181-182, Claimant testified that he was exposed to the dust generated by the materials he worked with continuously from 1973 until 1999. Tr. 132; see also

Tr. 180-81. Claimant's wife confirmed that the phosphate plant where he worked was dusty and dirty. Tr. 80-81. His clothes were covered in yellow dust that smelled like phosphorous when he came home from work, Tr. 87-89. He coughed up phlegm and black specs, and was short of breath. Tr. 81-82. Mrs. S. observed that Claimant has labored breathing and uses oxygen at home. Tr. 84-85. Employer provided no air quality test data in response to Claimant's discovery requests. Tr. 161-2.

In 1997, Claimant contracted tuberculosis. Tr. 133; 82. He believes he caught it from a crewman from a South American vessel who climbed the gantry to inquire about the tonnage of material Claimant had loaded onto the ship. Tr. 134-35.

Claimant's co-worker, Timothy Eustace, testified at the hearing. Tr. 173. He worked with Claimant since 1973, and served, at times, as a union official. Tr. 174. He confirmed that the terminal was a very dusty work environment, that the protection equipment provided was largely ineffective, and that the dust was so heavy it could be tasted through the masks. Tr. 176; Tr. 188. The parties stipulated that other scheduled co-worker witnesses would have testified to the same working conditions described by Eustice. Tr. 192.

Workplace Chemical and Other Exposures

In addition to the chemical exposure evidence produced by Claimant, his wife, and co-worker Eustice, Claimant's exhibit CX 17, pgs. 906 through 1154 provide the Material Safety Data Sheets (MSDS) for 266, but all, of the chemicals that were, from time to time, present at Claimant's worksites. CX Z is a 1984 report by the Phosphate Council that found, *inter alia*, that studied emphysema rates in phosphate workers.[4] Gene Mosca, Terminal Manager, now retired, testified at a deposition on January 18, 2006. CX 23. He confirmed that the workers at the terminal were chemical workers who were members of the International Chemical Workers Union. Dep. at 6. He also confirmed that the firebrick in the dryer, where claimant worked for a while, was replaced about every two years, and that there was asbestos in the dryer material. He testified further that ammonia, diesel fuel, and many other chemicals were present at the site. Dep at 16-23; 34-35; 47-49. He recalled that the respirators offered to the workers improved over time, Dep. at 25-

[4] Employer does not oppose Claimant's March 20, 2006, motion to admit the study as Claimant's exhibit CX Z, and claimant's motion is granted.

26, that EMSHA monitored exposure levels at the terminal, Dep. at 27; 44, and that the Employer tested certain workers' breathing. Dep. at 40-42.

Adverse Inference of Exposure

On August 1, 2003, Judge Stephen Purcell required Employer to produce documents in its possession or control covering the period of Claimant's employment, spanning thirty years including information covering, *inter alia*, Material Safety Data Sheets (MSDS) identifying the chemicals Employer used in its operations, its safety practices and policies relating to hazardous materials in the workplace, Employer's document retention policy, and information relating to litigation against Employer involving another former employee, Jimmy Johnson.

Employer failed to comply with that order, and as a consequence, I invoked, in an order issued on March 13, 2006, the adverse inference urged by Claimant that: "Employer's records would show that Claimant received excessive exposure to toxic chemicals and hazardous materials at work over a lengthy period of time, and that these exposures would support the diagnoses of his treating physician." That adverse inference shall hereinafter apply in this proceeding.

Tuberculosis

The CDC has reported that tuberculosis rates are significantly higher in South American countries than in the U.S. CX 18. Employer's terminal where Claimant worked loaded ships from around the world. CX 23, Dep. at 45. Claimant was exposed to a crewman from an allegedly South American vessel and later contracted tuberculosis.

Medical Evidence

Cardiac Claim

Dr. Joshua Furman, a specialist in internist medicine and cardiovascular disease, reviewed Claimant's medical records and evaluated his heart condition in a report dated January 19, 2006. CX 4. He noted that Claimant suffered a myocardial infarction (MI) on September 12, 1997, and underwent heart surgery on January 28, 1998. Thereafter, he engaged in a vigorous exercise program at home and at work. Dr. Furman continued: "the impact of dynamic aerobic exercise on the cardiovascular system is well documented in terms of its protective effect, myocardial conditioning against ischemic events, reduction in basic blood pressure

and heart rate, as well as impact on cardiovascular physiology including but not limited to endothelial function, vasodilation and myocardial oxygen consumption." It is undisputed that the work-related spinal injury on June 5, 1999, caused a decline in Claimant's ability to exercise, leading to a sedentary state, and this, in Dr. Furman's opinion, eliminated the advantages he received from the exercise and increased his risk of a cardiovascular event.

In addition, Dr. Furman noted that, as a result of the spinal injuries, Claimant received spinal interventions and anti-inflammatory medications, including Celebrex, from Drs. Castellvi and Dennison. From Dr. Ravi Khant he received Plavix, an antiplatelet drug, for his heart condition. Dr. Furman explained that to perform epidurals to treat Claimant's spinal injury, Dr. Dennison recommended suspension of the Plavix. Dr. Gandhi, then Claimant's cardiologist warned against that due to the increased risk of MI related to discontinuing Plavix. In Dr. Furman's opinion, the coronary events following both a gallbladder operation in August, 2004, and the epidurals in December, 2004, were "significantly affected" by the discontinuation of Plavix. He noted further that Claimant has not discontinued Plavix since then, and he has not had another event.

In Dr. Furman's opinion, the 1999 injury caused a "lack of activity" which "significantly contributed" to both cardiac events in 2004; the suspension of Plavix to facilitate the gallbladder surgery and the epidurals also "significantly contributed" to the myocardial injury from the two cardiac events in 2004; and Claimant's cardiac condition, in Dr. Furman's view, was permanently worsened as a result of the cardiac events which contribute to his ongoing need for cardiac care. CX 4.

Dr. Kenneth Neifeld, a specialist in Internal medicine, prepared a report dated January 17, 2006. CX 5. In it, he opined that:

1. A sedentary lifestyle caused by a back injury could cause or contribute to a worsening of a cardiac condition, and repeat myocardial infarctions would produce a permanently weakened cardiac state requiring ongoing care.
2. Long-term use of Celebrex "probably" contributed to the cardiac events in 2004. FDA labeling specifically warns of the risk of a cardiac event with long term use of Celebrex.
3. The discontinuation of Plavix before the epidurals "could cause and/or contribute to a subsequent myocardial infarction." CX 5.

Dr. Adithya Gandhi is Claimant's treating cardiologist. CX 8. On September 23, 2004, he recommended against stopping Plavix. Later, on December 1, 2004, he was again requested to authorize the suspension of Plavix to facilitate the epidural steroid injection therapy Dr. Dennison recommended for Claimant's spinal injuries. After expressing his reservations and fully informing Claimant of the cardiac risks of an MI, Dr. Gandhi provided the authorization. With the Plavix discontinued, Claimant suffered an MI on December 26, 2004. Thereafter, Dr. Gandhi informed Claimant that he would not again provide clearance to stop the Plavix. On April 18, 2005, Dr. Dennison suggested that an alternative to Plavix be prescribed so that Claimant could undergo surgery; however, Dr. Gandhi, on this occasion, refused to provide clearance to discontinue Plavix. CX 8.

Brandon Regional Hospital records show that Claimant was admitted for treatment following the MI he suffered after his gallbladder surgeryin Agust, 2004. A Discharge Summary dated August 9, 2004, reports that a catheterization revealed, *inter alia*, a total occlusion of the right coronary artery and of the vein graft to the first marginal: "which was successfully recanalized…." CX 24.

Tampa General Hospital records show that Claimant was admitted for treatment following the MI he suffered on December 26, 2004. CX 12. Dr. Cintron reported in the Hospital Discharge Summary that a catheterization on December 29, 2004, revealed a clot in the circumflex artery in the mid-distol location that was treated with angioplasty and stenting. Cx 12; *see also* Sullebarger, Dep, at 77. Hospital records indicate that the mid-circumflex artery clot was "most likely secondary to the patient stopping Plavix." Cx 12 at 489; CX 20.

Dr. Rauf Ordorica, in a report dated January 13, 2005, noted that a urinary obstruction procedure was deferred because Dr. Gandhi declined to authorize Claimant to again discontinue Plavix. CX 19.

Dr. John Sullebarger is a cardiologist who specializes in interventional cardiology. Dep. Ex 6. He examined Claimant on January 17, 2006, obtained his family history, smoking, work, and medical histories, and reviewed echocardiogram and EKG data. EX 2; Ex 6, Dep at 8. At the time, he noted that Claimant's ejection fraction, which is a measure of the heart's ability to pump, was 58% or slightly below normal of 60-plus. Dep at 12, 18. In his opinion, T.S. has high blood pressure and left ventricular hypertrophy (or thickening of the heart muscle), but not a severe disability. Dep at 13-17. He described the ejection fraction of 58% as a small problem indicative of minor damage. Dep at 18-19. Dr.

Sullebarger noted that since 1998, when Claimant's ejection fraction was in the low 40's, he has actually gotten better probably due to the medical care he received. Dep at 20-21.

Dr. Sullebarger observed that T.S. had progressive atherosclerotic disease in 2004, at the time he was taken off his antiplatlet drugs, and that posed a risk of triggering a thrombic event. He was also on Celebrex which, Dr. Sullebarger noted, has been shown to activate platelets and increase the risk of a thrombic event. Under these circumstances, Dr. Sullebarger explained that Claimant was the type of patient who presented the problem of caring for a cardiac patient who needs medical procedures, such as treatment for acute gallbladder, but runs the risks of an MI if his cardiac medication is discontinued. In Claimant's case, Dr. Sullebarger seemed satisfied that the risks were adequately weighed, but he explained that even though the event occurred, and it is not possible to quantify the "relative contribution of all those things." Dep. at 25-26, 29-30. Indeed, he observed that patients on Plavix can still have MI's, Dep at 28, 32, and patients who stop Plavix may not have an MI. For example, he noted that Claimant had stopped Plavix several times without incident. Dep at 32-33. With both Plavix and Celebrex, he considered it unusual for a patient to have an event in the absence of a blockage. Dep. at 30. He did acknowledge, however, that taking Celebrex or discontinuing Plavix may tip the balance. Dep at 30-31.

Dr. Sullebarger also addressed Claimant's contention that the decrease in the level of his physical activity contributed to his MIs. In Dr. Sullebarger's opinion, a lack of physical activity does not impact whether a patient gets blockages, and while Claimant's sedentary lifestyle is one factor along with diet and smoking habits and other factors, Dep. at 38, it: "is not a major factor in the generation of his atherosclerosis." Dep. at 37. Exercise, he reasoned, would not have a significant impact on stopping the progression of coronary artery disease. Dep at 39. Exercise has some benefit for the coronary system and the way the patient feels, but it does not, according to Dr. Sullebarger, impact the blockages themselves. Dep at 56.

Considering Claimant's current condition, Dr. Sullebarger opined that it is likely that Claimant's condition would have progressed whether or not he was taking Plavix or Celebrex. Dep at 36. He acknowledged that the MIs in 2004 may have resulted in damage, and, if so it is permanent; but the damage, if any, is "slight" and the impact on Claimant's lifestyle is, in Dr. Sullebarger's opinion, "trivial." Dep at 41. Dr. Sullebarger explained that the major cardiac event occurred in 1997, while the MIs thereafter were much smaller ones. Dep at 41. He

was, however, unable to conclude that cardiac events in 2002 and 2004 were the inevitable progression of the coronary artery disease, Dep at 42. To the contrary, Dr. Sullebarger thought that MIs happened on the days they did "because he had these procedures being done." Dep. at 43.

Reviewing Claimant's cardiac treatment, Dr. Sullebarger believed that Claimant has needed treatment since 1998 for blood pressure, cholesterol, blood sugar and atherosclerotic disease, and that continues. Dep at 45-6. He opined that Claimant would need the same care now even if the 1999 back injury had not occurred and he undergone none of the procedures or took none of the medications associated with that injury. Dep at 46-47. In summary, Dr. Sullebarger opined that the treatment and medical care Claimant now needs for his atherosclerosis was set in motion in 1997, Dep at 48, 69-70; however, he is unable to determine whether the damage seen in 2006 is the result of 1997 incident or the subsequent smaller ones. Dep at 44.

In terms of the two cardiac episodes in 2004, Dr. Sullebarger noted that Claimant's ejection fraction on a July, 2004, pre-operative stress test was 67% just before the gallbladder surgery. Dep. at 50-51. An ejection test in August, 2004, after the first of the two 2004 cardiac incidents was 60%. Dep. 66. After the second 2004, MI in December, 2004, his ejection fraction went down to 58%. Dep at 52. Dr. Sullebarger observed that the difference between an ejection fraction or 60 and 58 is probably within the margin of error for the test, Dep at 65, but 67 to 60 or 58 is definitely worse. Dep at 69-70. That, he explained, indicates a permanent worsening of the heart condition. Dep at 52. Each heart attack, Dr. Sullebarger noted, causes some permanent cell damage which is never replaced, Dep at 54-55, and the myocardial events Claimant has suffered contribute to his need for cardiac care.

Each event Claimant had was significant, but Dr. Sullebarger believes he still needs the same treatment. Dep. at 61. He acknowledged that Claimant had more visits to his cardiologist, Dep. at 73-4, but reviewing Claimant's medications, Dr. Sullebarger detected only temporary adjustments. Dep. at 72. He believes Claimant received the same long term care and treatment before and after the 2004 MIs. Dep. at 72. His records, however, went only until April of 2005, not to date. Dep. at 73.

After the August, 2004, MI, a catheterization revealed a total right occlusion that did not appear before, and required treatment different from the treatment Claimant received before the event. Dep. at 75. The catheterization on December

29, 2004, after the second MI in 2004, revealed a clot in the circumflex that was treated with angioplasty and stenting. Dep. at 77.

Pulmonary Claim

Dr. Richard England, a pulmonary disease specialist, prepared reports dated February 27, 2002; May 20, 2002, June 9, 2003, and July 30, 2004, EX 4, and was deposed on January 9, 2006. CX 22. In his May 20, 2002, report, Dr. England diagnosed occupational asthma based on Claimant's exposure to chemical dust and fumes in the workplace and the significant reversibility he detected on pulmonary function tests (PFTs). CX 22, Exh. C. In his June 9, 2003, and July 30, 2004, reports, he noted that Claimant has COPD, among other conditions, but he did not mention occupational asthma. EX. 4.

Dr. England first saw T.S. on April 4, 1997, and has been treating him ever since. Dep. at 4. He is familiar with Claimant's employment as a chemical worker, Dep. at 9, and in his opinion, Claimant has severe chronic lung disease, occupational asthma, and coronary artery disease. Dep. at 6. Although Dr. England had no specific knowledge of many of the specific chemicals present at the worksite, Dep. at 14, he attributed the occupational asthma to chemical exposures at work. Dep. at 7, 20. To make the diagnosis of occupational asthma, Dr. England explained that it would be helpful to know the specific substances, and amounts and frequency of exposure, Dep. at 23-25, and he further acknowledged that it is difficult to detect asthma in the presence of emphysema and COPD, Dep. at 28, but the factors that led him to conclude that occupational asthma was also present were exposure history, symptoms, and reversibility on PFTs. Dep. at 9-10, 18-19, 21-23.

By way of clarification, Dr. England's May 20, 2002, letter seemed to reference "occupational lung disease" and "occupational asthma" as two separate conditions, but he later made it clear that when he referred to the conditions "caused by occupational exposure," he was referring only to occupational asthma:

> Q. Okay, So, then, your opinion would be that of all the problems that he has, of which there appear to be many, the only one that you believe is related to occupational exposure would be asthma?
>
> A. Yes

Q. Occupational asthma?

A. That's correct. Dep. at 20.

Dr. England further considered asbestos exposure as a potential factor, historically, but he did not deem it significant medically. Dep. at 20, Dep. at 46. He also considered Claimant's exposure to tuberculosis, pneumonia, emphysema, fractured ribs, a 30 pack-year history of cigarette smoking, and coronary artery disease. Dep. at 26-27. In his opinion, however, the tuberculosis, pneumonia, and rib fracture, should not contribute to the long-term respiratory obstruction. Dep. at 27, 31-32. Tuberculosis, he noted, causes scarring and a restrictive defect. Dep. at 32. Dr. England deemed it impossible to tell where Claimant got the tuberculosis. Dep. at 46.

Focusing on the occupational asthma, Dr. England explained that it was indicated by reversibility, or improvement on the PFTs after the administration of bronchodilator medication. Asthma, he explained, is considered a reversible lung disease, whereas emphysema is not reversible. Dep. at 10, 28-29, 40. Dr. England interpreted the December 15, 2000, PFT as showing "significant improvement" with bronchodilators on the forced vital capacity and the FEV1, and demonstrated "probable reversibility;" however, the interpretation on the test itself indicated only a "mild response" to bronchodilators. Dep. at 40-41. Dr. England also cited the PFT conducted in May, 2002, during which Claimant went from an FEV1 of 1.41liters or 35%, of predicted for the patient's age, weight, and height before bronchodilators to 2.10 liters, or 52% of predicted, after bronchodilators. Dep. at 33-34.

Dr. England saw no indication of reversibility on the earlier tests, Dep. at 42, and in later years, the post-bronchodilator results were either insignificant or the tests declined after bronchodilators. Dep. at 35-36. Of the seven tests he reviewed, Dr. England thought two showed reversibility. Dep. at 42. He explained that, in some instances, the PFT results are influenced if patients take their aerosol before the test, because if they self-medicate before the test, the bronchodilator administered during the test will not work. Dep. at 37. As a result, patients are advised not to use their medication on the day of the test, but Dr. England explained that many are so well indoctrinated about taking their medication they are unable to break the habit. Dep. at 38. In this instance, however, there is no evidence establishing whether or not T.S. actually used his aerosol before any of these tests were conducted. Dep. at 37.

Finally, Dr. England testified that Claimant is on medication and nocturnal oxygen for his overall pulmonary condition, one element of which is exposure to chemical substances in the workplace. Dep. at 47.

Dr. Alan Goldman, a pulmonary disease specialist, examined Claimant on March 14, 2005, administered a PFT, reviewed his medical records and smoking histories, and thereafter prepared three reports, March 14, 2005; September 12, 2005, and December 20, 2005. EX 1; Dep. at 15.

The PFT results Dr. Goldman obtained showed;

Before bronchodilators:		After bronchodilators:
Vital capacity:	78%	82%
FEV1:	67%	71%

Arterial Blood gases: Po2 80; PCo2 42 Ph 7.39
Diffusing capacity: 51 %. Dep. at 18.

Dr. Goldman interpreted these data as showing mild obstructive and restrictive ventilatory defects, with mildly reduced diffusing capacity and normal blood gases. Dep. at 19. In his March 14, and December 20, 2005, reports, Dr. Goldman considered Claimant's smoking and medical histories, x-rays, CT scans, PFT, blood gas and other clinical data, symptoms, work, chemical exposure, and tuberculosis histories. Dr. Goldman assumed that Claimant had significant meaningful chemical exposure, Dep. at 70-72, and performed a physical examination. Based on all of the foregoing, he diagnosed, *inter alia*, bullous emphysema and COPD, but noted that he: "…did not see any evidence of any significant occupational lung disease…." EX 1.

In his deposition, Dr. Goldman testified that Claimant has bullous emphysema by CT scan, and Dr. Goldman attributes the emphysema to smoking. Dep. at 20, 30; 38-39. When asked whether he could separate the specific contributions to Claimant's COPD emphysema attributable to chemical exposures and smoking, Dr. Goldman explained that he could separate them because the chemical exposures did not cause Claimant's emphysema. Dep. at 38-39.

Commenting on Dr. England's opinion, Dr. Goldman noted that Dr. England diagnosed occupational asthma in 2002, at a time when he had PFTs from 1999, 2000, and 2001. As of that time, Dr. Goldman noted that Claimant's PFTs looked worse than they did more recently. Dep. at 25. In his opinion, the PFTs in 1996, 1997, and 1998, are almost the same as one administered in 2005, which showed a

mild impairment and no reversibility. Dep. at 42-43; 86. That suggests, Dr. Goldman opined, that the PFTs showing reversibility were either an aberration, Dep. at 25; 41-42, reflective of an acute problem or a testing error. Dep. at 26.

Addressing the question of reversibility, Dr. Goldman considered the possibility that Claimant could have both emphysema and asthma; but, in his judgment, the PFTs do not show the reversible asthma, because the tests which suggest reversibility are not consistent with either the ones administered before it or afterward. Dep. at 27-28. He explained that the pattern of reversibility over time is important in making a diagnosis. Dep. at 29. Eight PFTs were administered over a period of nearly ten years, and, Dr. Goldman noted that the 2000, 2001, and 2002 tests showed some reversibility on varying elements on the test. Dep. at 29-30. While he acknowledged that there are studies that show increased incidents of asthma in chemical workers, he noted further that Claimant was never hospitalized with Reactive Airways Dysfunction Syndrome and he shows no other evidence of occupational lung disease. Dep. at 72. Consequently, in Dr. Goldman's opinion, Claimant does not have asthma, and the data show no evidence of asthma or occupationally-induced lung disease. Dep. at 31-35; 65. Dr. Goldman did agree with Dr. England that the data do not support a diagnosis of asbestos-related lung disease. Dep. at 35; 65-66; 77-78.

Pulmonary Function Tests

Employer's Exhibit EX 4 includes PFTs. These show the following:

	Pre-bronchodilator	Post-bronchodilator
	October 20, 1996 Age 51 Ht.72" Wt.148	
FVC	4.21	Not performed
FEV1	3.12	Not performed
FEF 25/75	2.28	Not performed
Interpretation" "Normal spirometry"		
	December 4, 1997 Age 52 Ht.72" Wt.155	

FVC	4.11	4.11
FEV1	2.59	2.64
FEV1/FVC	63	64
FEF 25/75	1.31	1.44

Dr. England reported "No significant improvement after bronchodilators"

October 21, 1999
Age 54
Ht.72"
Wt.166

FVC	3.99	3.44
FEV1	2.43	2.46
FEV1/FVC	72	71
FEF 25/75	1.72	1.76

Dr. England reported "probable significant improvement after bronchodilators on FEF 25/75…No significant change pre and post FEV1"

December 15, 2000
Age 55
Ht.72"
Wt.166

FVC	3.40	3.90
FEV1	2.32	2.62
FEV1/FVC	68	67
FEF 25/75	1.41	1.50

Dr. England reported significant improvement with bronchodilators on FVC and FEV1

May 3, 2001
Age 55 (sic)
Ht.72"
Wt.166

FVC	2.59	3.03
FEV1	1.41	2.10
FEV1/FVC	54	69
FEF 25/75	0.55	1.51

Dr. England reported significant improvement in the FEV1 and FEV1/FVC ratio with bronchodilators. Computerized "Interpretation" noted a "good response to bronchodilators."

September 12, 2002
Age 57
Ht.72”
Wt.195

FVC	2.92	3.13
FEV1	1.75	1.84
FEV1/FVC	60	59
FEF 25/75	1.08	1.15

Dr. England noted a “significant improvement” on the FEF 25/75 after bronchodilators, but the computerized interpretation of the FVC showed “an insignificant response to bronchodilators” on that aspect of the test.

January 22, 2004
Age 58
Ht.73”
Wt.172

FVC	3.32	3.31
FEV1	1.93	1.78
FEV1/FVC	58	54
FEF 25/75	1.03	0.71

Dr. England noted “no significant improvement” after bronchodilators.

July 29, 2004
Age 59
Ht.72”
Wt.170

FVC	3.13	3.25
FEV1	2.10	1.55
FEV1/FVC	67	48
FEF 25/75	1.48	0.99

Dr. England noted “no significant improvement” after bronchodilators.

Discussion

The record shows that Claimant is currently receiving permanent total disability compensation as a consequence of the work-related cervical spine injury he suffered on June 5, 1999. *See*, *T.F.S.* v. IMC-Agrico MP, Inc., 2002 LHC 00573 (ALJ Oct. 31, 2003). In this proceeding, he claims medical benefits for both a cardiac condition allegedly arising out of the physical restrictions and medical treatments necessitated by the back injury and for a pulmonary condition allegedly

caused by years of exposure to the miasma of chemical fumes and dusts which permeated the Employer's worksite. The cardiac and pulmonary claims were consolidated for hearing, and are considered below.

Section 20 Presumption

The record shows that Claimant suffered two heart attacks in 2004, and Claimant argues that he is entitled to rely upon the presumption in Section 20 of the Act to establish that these cardiac events were related to the 1999 cervical injury at work. He also insists that he suffers from pulmonary conditions, including occupational asthma, and that the presumption applies, as well, to these conditions. Employer responds that the cardiac claim is not aided by the presumption. It contends that Claimant has advanced pre-existing coronary artery disease, suffered heart attacks in 1997 and 2002, and had open heart surgery and numerous catheterizations and stents before the two cardiac events in 2004. Accordingly, as Employer sees it, it is "impossible" to determine that Claimant's 2004 heart attacks were caused by his back treatment. It, therefore, concludes that "the §20 presumption does not apply." Emp. Br at 9.

Addressing the pulmonary claim, Employer argues that Claimant has chronic obstructive pulmonary disease (COPD) including emphysema, but it is caused by 30 years of smoking, a bout with tuberculosis, and lung punctures unrelated to chemical exposures in the workplace. Employer does not specifically assert that the presumption is inapplicable to the pulmonary claim, Emp. Br. at 16-20, but argues that the medical evidence establishes that Claimant has no occupational-related pulmonary disease.

Before turning to the individual claims, it should be noted that the courts have held that the Section 20(a) presumption is applicable to medical benefits. Jenkins v. Maryland Shipbuilding & Dry Dock Co., 6 BRBS 550 (1977), rev'd on other grounds, 594 F.2d 404, (4th Cir**.** 1979). Moreover, it can be triggered by credible testimony that an incident occurred or conditions existed at work that could have precipitated the injury. Conoco, Inc. v. Director, 33 BRBS 187 (CRT) (1999); Damiano v. Global Terminal & Container Service, 32 BRBS 261 (1998); Marinelli v. American Stevedoring, Ltd, 34 BRBS 112 (2000). In this instance, there is credible medical evidence provided by Dr. Furman and by Dr. Neifeld that Claimant's sedentary lifestyle, necessitated by the physical restrictions caused by the 1999 cervical injury at work, and the Celebrex prescribed to treat that injury contributed to the heart attacks Claimant suffered in August and December of 2004. In addition, Tampa General Hospital records, and Dr. Furman attributed the

December, 2004, heart attack to the discontinuation of the heart medication, Plavix, which was necessary in order to permit the epidural steroid injection that were administered to treat Claimant's cervical injuries.

Under these circumstances, credible medical evidence has, therefore, been adduced that Claimant's work-related sedentary lifestyle and the medication, Celebrex, contributed to both heart attacks. In addition, credible medical evidence also has been adduced that medical preparations necessary to permit the epidural injection treatment of Claimant's cervical injuries, i.e., the temporary suspension of Plavix, caused the December 26, 2004, cardiac event. As such, the presumption applies to both 2004 thrombic events.[5]

Claimant also argues that his pulmonary condition is due to tuberculosis, emphysema, and occupational asthma, all of which he alleges are attributable to his work. Again, the evidence is clearly sufficient to invoke the presumption that Claimant's pulmonary condition is, at least in part, causally related to the chemical

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[5] Contending that it is "medically impossible" to determine that Claimant's back injury treatments caused his heart attacks, Employer argues that Claimant cannot invoke the presumption in Section 20, and cites Director v. Greenwich Collieries, 512 U.S. 267 (1994) in support of its assertion. Employer misapplies Greenwich Collieries. In Greenwich Collieries, the Supreme Court struck down administrative rulings under the Black Lung Act that required the invocation of a presumption in favor of claimants in circumstances in which the evidence relating to an element necessary to trigger the presumption, such as the existence of pneumoconiosis, was in equipoise and "true doubt" existed in the record. Under the applicable precedents at the time, if the presumption was invoked by evidence showing the existence of pneumoconiosis, for example, it could not then be rebutted by evidence which would demonstrate the absence of pneumoconiosis. Under these circumstances, the Court ruled that the Administrative Procedure Act, imposed the burden of proof upon claimants, and, accordingly, when the evidence is in equipoise and "true doubt" exists, that burden has not been satisfied.

The presumption in Section 20 of the Longshore Act does not work the same way as the presumption the Court addressed in Greenwich Collieries. As the Board has held, the prima facie elements a claimant must satisfy to invoke the Section 20(a) presumption include evidence of injury, i.e., harm, and working conditions that could have caused the harm. Care v. Washington Metropolitan Area Transit Authority, 21 BRBS 248 (1988). In order to invoke Section 20(a), claimant is not required to introduce affirmative medical evidence establishing that the working conditions in fact caused the alleged harm. In Sinclair v. United Food & Commercial Workers, 23 BRBS 148 (1989), the Board explained that it is sufficient to invoke the presumption if the relevant medical opinions indicate a possible connection between claimant's symptoms and the employment-related exposure to chemicals. In O'Kelley v. Dep't of the Army/NAF, 34 BRBS 39 (2000), for example, the opinions of two physicians that pesticide exposure could have caused or aggravated claimant's neurological symptoms was sufficient to invoke the presumption. The cases demonstrate that to invoke the Section 20(a) presumption, claimant is not required to introduce affirmative evidence establishing that the working conditions, in fact, caused the alleged harm. Rather, claimant's burden is to establish the existence of working conditions that could have caused the harm. Stevens v. Tacoma Boatbuilding Co., 23 BRBS 191 (1990); *see also*, Bath Iron Works v. Brown, 194 F.3d 1, (1st Cir. 1999); Hargrove v. Strachan Shipping Co., 32 BRBS 11, aff'd on recon., 32 BRBS 224 (1998).

Once claimant shows physical harm and a work-related accident which could have caused the harm, the Section 20(a) presumption applies; and it is employer's burden to rebut the presumption. It may do so by 'ruling out' a causal link in the Eleventh Circuit or by adducing substantial evidence severing the causal link in other circuits. When employer produces such evidence, the presumption drops out of the case; and the record as a whole must be weighed to determine whether claimant has satisfied his burden of proof in establishing causation. MacDonald v. Trailer Marine Transport Corp., 18 BRBS 259 (1986), aff'd mem. sub nom. Trailer Marine Transport Corp. v. Benefits Review Board, 819 F.2d 1148 (11th Cir. 1987). If there is "true doubt" about whether that burden has been met or if the evidence is in equipoise, Claimant can not prevail. Maher Terminals, Inc. v. Director, 992 F.2d 1277(3d Cir. 1993), aff'd sub nom. Director v. Greenwich Collieries, *supra*. This is the way the way the section 20 presumption is applied, and the Board has thus held that the Supreme Court's decision in Greenwich Collieries does not change or affect the law regarding invocation and rebuttal of the Section 20(a) presumption. Holmes v. Universal Maritime Service Corp., 29 BRBS 18 (1995) (Decision on Recon.).

dusts and fumes to which he was exposed during the 30 years he worked for the employer.

Claimant testified that chemical dusts and fumes permeated his work environment for many years exposing him to a host of hazardous materials against which the protective gear furnished by Employer was largely ineffective. Claimant's testimony was substantially corroborated by his co-worker, Tim Eustace, and by a former terminal manager, Gene Mosca, by the pile of MSDS's in evidence supplied by the Employer which identify some, but not all, of the materials which, from time to time, were found at the terminal, and by the report of the Phosphate Council. In addition, the adverse inference entered in this matter establishes that Employer's records which were not produced in discovery would show that "Claimant received excessive exposure to toxic chemicals and hazardous materials at work over a lengthy period of time, and that these exposures would support the diagnoses of his treating physician."

Under such circumstances, the Board has held that a Claimant's credible testimony that "working conditions" existed that could have caused injury is sufficient to establish that element of his prima facie case. Quinones v. H.B. Zachery, Inc., 32 BRBS 6 (1998), rev'd on other grounds, 206 F.3d 474, (5th Cir. 2000). Claimant has thus demonstrated working conditions sufficient to invoke the presumption that his pulmonary disease is, partially, attributable to his employment.

While Dr. England, Claimant's treating pulmonary specialist, did not believe his emphysema was work related, he did, in part, support Claimant's case. Dr. England considered Claimant's exposure history, symptoms, clinical tests, including x-rays and pulmonary function tests (PFT) data, and examination results, diagnosed occupational asthma. In addition, as a consequence of Employer's non-compliance with a discovery order, and for the reasons set forth in the Order issued on March 13, 2006, an adverse inference was entered in this matter that Employer's records would show that "Claimant received excessive exposure to toxic chemicals and hazardous materials at work over a lengthy period of time, and that these exposures would support the diagnoses of his treating physician." Considering Dr. England's expert opinion and the adverse inference that supports it, the evidence is sufficient to trigger the presumption in Section 20 of the Act that Claimant has occupational asthma, a pulmonary condition arising out of chemical dust and fume exposures at his place of work.[6]

[6] In Peterson v. Columbia Marine Lines, 21 BRBS 299 (1988), for example, the Board rejects the argument that because a doctor was unable to identify the specific chemicals which produced claimant's chemical hypersensitivity,

Tuberculosis

Claimant also argues that his work is responsible for the tuberculosis he contracted. Claimant has placed in evidence a CDC study documenting higher incidents of tuberculosis in South American countries than in the U.S., and he testified about an incident at work involving a crew member of a South American vessel who approached him on the gantry to inquire about the quantity of materials being loaded aboard the ship. Claimant referred the crewman to his supervisor, but asserts that this encounter was sufficient to trigger the presumption that the tuberculosis was contracted from this South American crewman and was, therefore, work-related. Yet, the scenario Claimant weaves about this incident is speculation predicated upon speculation, and it is insufficient to trigger the presumption.

Although Claimant believes the vessel was South American, the record shows that vessels from around the world loaded and unloaded at the terminal; and it does not show the name or registry of the vessel or that it was South American. Moreover, even if it is assumed that the vessel was South American, the record does not show the nationality of the particular crewman who visited Claimant on the gantry. Many such vessels are manned by polyglot crews, rendering assumptions about national origin problematic. Nor is there any evidence that the crewman or anyone else aboard the ship actually had or carried tuberculosis or that the supervisor or anyone else the crewman spoke with tested positive. Indeed, the only other positive TB test result reflected in this record was produced by Claimant's wife. Claimant has assumed that her exposure emanated from his exposure to the crewman; but this record leaves open the epidemiological potential that her exposure may have preceded his. In summary, there is speculation, but no credible evidence, that Claimant was exposed to tuberculosis on the job.[7] The mere fact that Claimant spoke at work with a crewman from a vessel believed to be from South America is, alone, not sufficient to trigger the presumption of etiology of Claimant's tuberculosis.

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his opinion was insufficient to support a finding of causation, where the doctor stated that claimant's symptoms were due to the cumulative effect of chemical exposures over many years and that any or all of the chemicals to which he was exposed could have played a part in his symptomatology.

[7] Claimant was also exposed to tuberculosis, Tuberculosis causes scarring and a restrictive defect. Dep. at 32. Dr. England testified that: "It's not possible to tell where he got the tuberculosis." Dep. at 46.

Rebuttal

Once the presumption regarding the work-related etiologies for Claimant's cardiac condition, pulmonary conditions is invoked, it is Employer's burden within the jurisdiction of the Eleventh Circuit Court of Appeals, where this case arises, to rule out a causal relationship between the employment and the injury. Brown v. Jacksonville Shipyard, 893 F.2d 294 (11th Cir. 1990); *Contra*, Conoco v. Director, 194 F. 3d 684 (5th Cir. 1999) (requiring employer to go forward with substantial countervailing evidence to rebut the presumption). *See also*, Merill v. Todd Pacific Shipyards Corporation., 25 BRBS 140 (1991), *aff'd*, 892 F.2d 173, 23 BRBS 12 (CRT) (2d Cir. 1989). In determining whether Employer has ruled out a causal relationship between Claimant's employment and the injury as required by Brown v. Jacksonville Shipyard; however, it is necessary to consider the entire record, not just substantial countervailing evidence that would tend to sever the causal nexus. Thus, it appears that the analysis required to rule out a work-related etiology is quite similar to the analysis required in other Circuits after the presumption has been triggered and rebutted. *See,* Del Vecchio v. Bowers, 196 U.S. 280 (1935). It would seem to require an analysis which takes into consideration the record evidence viewed in its entirety. Applying Brown v. Jacksonville Shipyard, we turn first to the pulmonary claim and then address the cardiac condition.

Pulmonary Conditions

Emphysema and COPD

As noted above, Claimant has triggered the presumption that, except for the residuals of tuberculosis, his pulmonary disease is related to his work at Employer's terminal. Dr. Goldman, however, examined Claimant, reviewed his chemical exposure history, and the clinical data, and concluded that Claimant's pulmonary condition is not related to his work. In this respect, he disagrees with Claimant's treating physician, and before it can be determined whether Dr. Goldman's opinion rules out a nexus between Claimant's work and his pulmonary condition, it is first necessary to set forth precisely what Dr. England diagnosed and his assessment of the etiologies of the conditions he diagnosed.

The record shows that Dr. England was aware of Claimant's 30 year smoking history and his lengthy exposures to significant amounts of chemicals including, among other chemicals, phosphates, diesel fumes, PCBs, and ammonia in the workplace. Dr. England diagnosed occupational asthma, emphysema, and

COPD. He explained further that the emphysema and COPD are confirmed diagnoses, separate from the diagnosis of occupational asthma, and he noted that tuberculosis, pneumonia, and a rib fracture should not contribute to the long-term obstruction. Moreover, although the Phosphate Council study reported a higher incidence of emphysema in chemical workers, Dr. England made clear that he concluded that the only pulmonary condition Claimant has that he attributes to occupational exposure is the occupational asthma.

Claimant argues that Dr. England's rejection of chemical exposure as cause of his emphysema should be rejected because he was unaware of the adverse inference on exposure that supported his diagnosis; and argues further that the opinions of both Dr. England and Dr. Goldman which attribute his emphysema to smoking not chemical exposure should be rejected. Claimant notes that the Phosphate Council study was not produced until after the trial and, as a consequence, neither doctor was aware of the study which, according to Claimant, shows an increased incident of emphysema in phosphate workers. Cl. Br. at 32-33. It is true that Drs. Goldman and England did not have monitoring data to show how high the exposure levels were, but they did have Claimant's own reported history of exposure which described high levels of exposure to the chemical dusts and fumes.

Moreover, while the Phosphate Council study provided general survey data and general conclusions, with significant limitations that will be addressed in a moment, Dr. England and Dr. Goldman evaluated Claimant's specific conditions and the specific etiologies associated with his particular pulmonary presentations. To be sure, the presumption of etiology was triggered based upon Claimant's testimony of exposure and the environmental conditions at the terminal; however, in rebuttal, it is relevant that Dr. England agrees with Dr. Goldman that Claimant's COPD and emphysema are not attributable to the chemical exposures Claimant experienced at work. I am, of course, mindful that Dr. England did not attribute Claimant's emphysema and COPD to chemical exposures, but neither did he specifically rule out chemical exposure as cause of emphysema or COPD in accordance with Brown v. Jacksonville Shipyard. Dr. Goldman, however, did rule out a causal link, and his opinion is not refuted by any medical evidence in this record. In his opinion Claimant's emphysema and COPD are probably due to smoking. While the smoking attribution may be deemed equivocal, Dr. Goldman unequivocally de-linked chemical exposure as an etiology for Claimant's COPD emphysema, and this assessment by Dr. Goldman, directly and expressly, severs that presumptive causal link. Moreover, Claimant's pulmonary treating physician,

who is most familiar with his emphysema and COPD, rather than refuting Dr. Goldman's conclusion, essentially agreed with him.

Claimant next contends that Dr. England's rejection of chemical exposure as cause of his emphysema should be rejected because he was unaware of the adverse inference on exposure that supported his diagnosis, and argues further that the opinions of both Dr. England and Dr. Goldman that the emphysema was caused by smoking, not chemical exposure, should be rejected. Claimant notes that the Phosphate Council study was not produced until after the trial and, as a consequence, neither doctor was aware of the study results which, according to Claimant, showed an increased incident of emphysema in phosphate workers. Cl. Br. at 32-33. Claimant, however, misreads both the inference and the Phosphate Council's epidemiological study.

The adverse inference Claimant sought, and the Order which issued on March 13, 2006, imposed in this proceeding, immunized Dr. England's opinions from the Employer's challenge that his diagnoses were flawed because he did not know all of the chemical exposures Claimant actually experienced during the course of his employment. The adverse inference established that: "Claimant received excessive exposure to toxic chemicals and hazardous materials at work over a lengthy period of time, and that these exposures would support the diagnoses of his treating physician." The inference did not add to the totality of the information Dr. England had relating to the etiology of Claimant's emphysema. What he used as basis for his evaluation when he formulated his diagnoses was not altered by the inference. He was aware of Claimant's smoking history and Claimant's description of his work and exposure histories and knew that Claimant had high levels of exposure to chemical dusts and fumes. He, therefore, had sufficient information to attribute the etiology of Claimant's emphysema to chemical exposure if he considered it the cause, but he did not. *See, e.g.*, Peterson v. Columbia Marine Lines, *supra*, fn. 2. The adverse inference did not change or add to that circumstance.

Nor do the results of the Phosphate Council report warrant rejection of the etiology assessment provided by Drs. Goldman or England. A careful reading of the epidemiological study sponsored by the Phosphate Council reveals that it does not conclude, as Claimant contends, that phosphate workers generally showed an increased incident of emphysema. To the contrary, the study clearly defines several limitations that refute Claimant's contention that its results apply to him.

The study, for example, found an association with emphysema in white males for the sub-cohort job category defined as "Mining and Beneficiation" (the process of washing, sizing, and floatation of ore to remove sand and clay); however, the beneficiation jobs were "apparently unrelated to emphysema," CX Z at 86, and Claimant was not a miner. In addition, Rock Processing showed increased risk of mortality, but Rock Storage and Drying and Rock Grinding "were unrelated to the relative risk of emphysema." Id. Thus, the types of work Claimant performed in storage and drying do not seem to relate to increased rates of emphysema. Moreover, even these data have limited application to Claimant.

The Phosphate Council report authors specifically noted that: "This study was restricted to mortality, therefore, an evaluation of non-fatal health effects was precluded." CX Z at v. Further, Claimant has a 30-year smoking history, and the study report was careful to caveat that: "… the absence of valid information on cigarette smoking, which is the principal non-occupational determinant of lung cancer and emphysema, prevents us from determining whether the results of the study were confounded by the smoking habits of workers." Id.

In summary, it appears that the Phosphate Council study is not especially pertinent to Claimant's situation, but beyond that, the Board has held that regardless of the absence of a definitive study regarding the relationship between certain chemicals and a claimant's ailment, the opinions of physicians that the ailment is not related to his exposure to hazardous chemicals are a result of their professional assessment regarding the cause of claimant's injury and, therefore, are adequate to constitute specific and comprehensive evidence sufficient to rebut the presumption. Devine v. Atlantic Container Lines, G.I.E., 23 BRBS 279 (1990). Accordingly, I conclude that the presumption applicable to Claimant's COPD and emphysema is rebutted. Considering the record as a whole, including the Phosphate Council study, the medical evidence relating specifically to Claimant's pulmonary condition, rules out chemical exposure in the workplace as a cause contributing to his emphysema or COPD, and thus rebuts the presumption in compliance with Brown v. Jacksonville Shipyard. *See*, Phillips v. Newport News Shipbuilding & Dry Dock Co., 22 BRBS 94 (1988)(breathing disorder not causally related to asbestos exposure where medical evidence indicated that the claimant had a severe chronic obstructive pulmonary disease which was caused by prolonged cigarette smoking).

Occupational Asthma

The record is quite different as it relates to the diagnosis of occupational asthma. On May 20, 2002, Dr. England prepared a report. In it, he diagnosed

occupational asthma related to chemical exposure in the workplace. Dr. Goldman, in contrast, concluded that Claimant does not have occupational asthma, and the issue is whether Dr. Goldman's unequivocal opinion, in the context of the record considered as a whole, is sufficient to rule out the presence of the work-related ailment. For the reasons which follow, I find and conclude that Dr. Goldman's analysis does rule out a causal link and is sufficient to rebut the presumption.

Dr. England first saw T.S. on April 4, 1997, and has been treating him ever since. At his deposition he testified that three factors led him to conclude that occupational asthma was present: (1) exposure history, (2) symptoms, and (3) reversibility on PFTs after the administration of bronchodilator medication. He acknowledged that occupational asthma was a difficult diagnosis to make, and he could not confirm it absolutely; but that sufficient reversibility was demonstrated to conclude that exposure to chemicals at work caused Claimant's occupational asthma. Diagnostically, the occupational asthma was indicated, in his opinion, by improvement detected on post-bronchodilator PFTs, because, he reasoned, asthma is considered a reversible lung disease while emphysema is not reversible. Yet the PFT data was mixed.

The record shows that a PFT administered on October 20, 1996 was interpreted as a "Normal spirometry." The December 4, 1997, PFT, as interpreted by Dr. England showed "No significant improvement after bronchodilators." Reviewing the results of an October 21, 1999, PFT, Dr. England reported "probable significant improvement after bronchodilators on FEF 25/75…No significant change pre and post FEV1." At his deposition, however, Dr. England indicated that no reversibility was shown on the 1997 or 1999 test. Dep. at 42. Dr. England did note significant improvement in the FVC and FEV1 with bronchodilators on the December 15, 2000, PFT; however, at his deposition, he testified that this test demonstrated "probable reversibility," and he acknowledged that the interpretation on the test itself indicated only a "mild response" to bronchodilators.

Sixteen months later, Dr. England interpreted the May 3, 2001, PFT as showing a significant improvement in the FEV1 and FEV1/FVC ratio with bronchodilators. On the September 12, 2002, PFT, he noted a "significant improvement" on the FEF 25/75 after bronchodilators, but the interpretation provided on the test report itself stated that the FVC showed "an insignificant response to bronchodilators" on that aspect of the test. Thereafter, Dr. England interpreted the January 22, 2004, and the July 29, 2004, PFTs as showing "no significant improvement" after bronchodilators.

In summary, of the seven tests he reviewed, Dr. England testified at his deposition that he thought two showed reversibility, (Dep. at 42); the December 15, 2000, PFT and the May 3, 2002, PFT. He agreed that no reversibility was demonstrated on the 1997 or the 1999 test, and that the most recent tests showed no reversibility after bronchodilators. He explained that the asthma may be intermittent or variable, and, in some instances, the PFT results may be distorted if the patient has taken his breathing medication before the test, thus diminishing the effectiveness of the bronchodilator administered during the test. He testified that patients are advised not to use their medication on the day of the test, but many do anyway. He acknowledged, however, that he was unable to determine whether Claimant had actually used his aerosol medication before any these tests were conducted, and Claimant could not specifically recall whether he had or not.

Dr. Alan Goldman prepared reports dated September 12, 2005, March 14, 2005, and December 20, 2005, and was deposed on January 18, 2006. In his March 14, 2005, and December 20, 2005, reports, he considered Claimant's smoking and medical histories, x-rays, CT scans, PFTs, blood gas and other clinical data, symptoms, work, and tuberculosis histories. In addition, he performed a physical examination, and assumed that Claimant had a significant, meaningful chemical exposure history. He diagnosed, *inter alia*, bullous emphysema and COPD; but, significantly, he reported that he: "…did not see any evidence of any significant occupational lung disease…."

During the visit on March 14, 2005, Dr. Goldman obtained PFT and blood gas data. The results were as follows:

	Pre-bronchodilators:	Post-bronchodilators:
Vital capacity:	78%	82%
FEV1:	67%	71%

Arterial Blood gases: Po2, 80; PCo2, 42; Ph 7.39; Diffusing capacity: 51 %.

Dr. Goldman interpreted these data as showing mild obstructive and restrictive ventilatory defects with mildly reduced diffusing capacity and normal blood gases. He diagnosed bullous emphysema by CT scan, probably due to smoking. Commenting on Dr. England's diagnostic methodology for detecting occupational asthma, Dr. Goldman noted that as of 2002, when Dr. England opined in his May 20, 2002 letter that Claimant had occupational asthma, Dr. England had PFTs prior to 2002. Comparing those data with other PFT studies, Dr. Goldman noted that Claimant's PFTs looked worse then than they did more recently. He

observed that the PFT results in 1996, 1997, and 1998, are almost the same as the results obtained on the 2005 PFT which he interpreted as showing a mild impairment and no reversibility. These results, in Dr. Goldman's opinion, indicate that the PFTs that showed reversibility were aberrational in light of the earlier and later studies that showed no reversibility.

Addressing the question of reversibility in more detail, Dr. Goldman noted that in differentiating emphysema from asthma, the pattern of reversibility over time is important in making a diagnosis. In this instance, the pattern of the PFT data over time does not, in Dr. Goldman's opinion, evidence reversible asthma, because, despite the variability of asthma, the data indicating reversibility are sandwiched between the early PFTs which reveal no reversibility and the most recent PFTs which also show no reversibility. Under these circumstances, Dr. Goldman found that the two PFTs that revealed reversibility were outliers the may have been invalid or indicative of a temporary, acute ailment that responded to the bronchodilators.

Dr. Goldman acknowledged that there are studies indicating increased incidents of asthma in chemical workers; however, he noted that Claimant was never hospitalized with Reactive Airways Dysfunction Syndrome and shows no other evidence of occupational lung disease. Consequently, since the data specific to Claimant, in his opinion, show no evidence of asthma or occupationally-induced lung disease, he concluded that Claimant does not have asthma. Finally, Dr. Goldman opined that going from a very active to a very sedentary lifestyle would not contribute to a respiratory impairment, and he agreed with Dr. England that the data do not support a diagnosis of asbestos-related lung disease.

Weighing Conflicting Medical Opinions

In evaluating the conflicting opinions of Drs. England and Goldman regarding the diagnosis of occupational asthma, I have taken into account that both are highly qualified pulmonary specialists. I have also accorded substantial weight to the fact that Dr. England is Claimant's treating physician. Nevertheless, the analyses and conclusions provided by Dr. Goldman are well-reasoned and appear better supported by the clinical data than the contrary opinion of Dr. England; particularly with regard to the pulmonary function data addressing the question of reversibility which Dr. England relied upon to differentiate emphysema from asthma. As Dr. England explained, he was able to detect and diagnose the occupational asthma in May of 2002, because it revealed itself as a reversible impairment on the post-bronchodilator PFT.

Dr. Goldman, however, reasoned that the PFT data as a whole demonstrate that the particular data Dr. England used to diagnose asthma were not indicative of a chronic asthmatic condition. He noted that the PFT studies in 1996, 1997 and 1998, administered before Claimant ceased working due to the 1999 cervical spine injury revealed no reversibility. Similarly, PFTs in 2002, 2004, and 2005, revealed no significant reversibility. As a consequence of this pattern of data, Dr. Goldman considered the two tests that indicated reversibility aberrations that were either invalid or indicative of an acute condition, not occupational asthma.

I am mindful, that Dr. England, in turn, suggested that the tests that revealed no significant reversibility were the invalid tests. At his deposition, he reviewed the most recent tests and testified that they do not persuade him to change his opinion, observing instead that the absence of reversibility on these tests could be explained if the patient, contrary to express pre-test instructions, used an aerosol medication before the taking the test, thereby rendering the post-bronchodilator results invalid. While I do not lightly dismiss the notion that patients sometimes act contrary to the instructions they receive, I am not, in the absence of evidence to the contrary, persuaded that Claimant twice followed those instructions but disregarded them on six other occasions. Moreover, in the absence of evidence supporting the invalidity of the six PFTs that revealed no significant reversibility, Dr. England has otherwise failed to address the significance of the pattern of PFT data as it relates to the question of reversibility. He did not, for example, explain or address how the pattern of data showing no reversibility on early PFTs, followed by two tests showing reversibility, followed by four tests over a three year period which reverted back to revealing no significant reversibility factored into his 2002 diagnosis of reversible occupational asthma.

As a result, while the adverse inference of exposure supports Dr. England's diagnosis of occupational asthma, the principal diagnostic indicators he employed to formulate that diagnosis were outlier instances of impairment reversibility which the totality of the pulmonary function data do not support. In contrast, Dr. Goldman specifically relied upon the pattern of reversibility data to conclude that it did not reveal the existence of occupational asthma; and except for the suggestion that the recent tests may be invalid; a premise not supported by the record, Dr. England did not otherwise refute Dr. Goldman's analysis.[8]

[8] Notably, neither Dr. Goldman nor Dr. England assessed, on this record, the validity of any of the PFT's based upon a review of the smoothness of the slopes on the graph tracings these tests produce or the consistencies or inconsistencies in the graph tracings among the various attempts on each test.

For these reasons, then, I find and conclude that Dr. Goldman's opinion is supported by the clinical data and is better reasoned than the contrary opinion of Dr. England. Reversibility was clearly not the only factor Dr. England considered, but he surely identified it as the key factor he relied upon to diagnose occupational asthma. Yet, the pattern of reversibility, over time, including the most recent PFTs, as Dr. Goldman explained, do not support a diagnosis of occupational asthma. Thus Dr. Goldman has provided an analysis that overcomes the treating physician's opinion as supported by the adverse inference applied in this matter. For all of the foregoing reasons, I have accorded greater weight to Dr. Goldman's opinion than the contrary medical opinions in this record, and Dr. Goldman has, based upon the clinical data, unequivocally ruled out occupational asthma as a pulmonary disease contributing to Claimant's condition. *See, e.g.*, Phillips v. Newport News Shipbuilding & Dry Dock Co., 22 BRBS 94 (1988); *See also*, O'Berry v. Jacksonville Shipyards, 21 BRBS 355 (1988)(presumption rebutted in a claim for asbestosis where claimant's lung condition was siderosis and not asbestosis).

Accordingly, I conclude that Employer has rebutted the presumption that Claimant has an occupationally related respiratory or pulmonary condition not only under Brown v. Jacksonville Shipyard, but in accordance with rebuttal criteria applied in other circuits. *See*, Ortco Contractors, Inc. v. Charpentier, 332 F.3d 283, (5th Cir. 2003), cert. denied, 124 S. Ct. 825 (2003); Bath Iron Works Corp. v. Director, 137 F.3d 673, (1st Cir. 1998). Considering the evidence reviewed in its entirety, Claimant has failed to establish that chemical exposures in the workplace caused or contributed to his current pulmonary conditions. His pulmonary claim must, therefore, be denied. Universal Maritime v. Moore, 126 F.2d 256 (4th Cir. 1997); O'Kelley v. Dep't of the Army/NAF, 34 BRBS 39 (2000).

Cardiac Claim

Claimant's consolidated claim also seeks medical benefits for his cardiac condition. The record shows that has severe pre-existing coronary artery disease and has suffered several heart attacks. The two most recent cardiac events were in 2004; the first one on August 9, 2004, followed surgery for a gallbladder condition, and the second on December 26, 2004, occurred after epidural steroid injections for the 1999 work-related back injury. Claimant alleged that these two heart attacks were attributable to his 1999 cervical spine injury because that injury caused him to give up the exercise program he had undertaken to improve his condition following his two heart attacks in 1997 and 2002, respectively, and required him to take Celebrex, a prescription medication which FDA has warned increases the risk

of heart attack. He also contended that the last heart attack that followed the epidurals in December, 2004, was a result of these factors, but also the fact that he had to discontinue taking his heart medication, Plavix, in order to undergo the epidurals. Suspension of the Plavix, Claimant alleged, was an additional contributing cause of the December 26, 2004, thrombic event. *See*, Cl. Br. at 17-20; Cl. Rply in Support of Mot. to Strike at 2-4. As such, citing Independent Stevedores v. O'Leary, 357 F.2d 812 (9th Cir. 1966) and Rajotte v. General Dynamics, 18 BRBS 85 (1986), Claimant averred that the 1999 injury at work contributed to, combined with, and aggravated his pre-existing coronary artery disease and heart condition; and as such, the entire resultant disability is compensable.

Employer responded that Claimant's heart attacks were due to his pre-existing coronary artery disease, and it disputed the notion that Claimant's sedentary lifestyle, his Celebrex medication, or the suspension of Plavix associated with the epidural steroid injections administered in December, 2004, had any demonstrable effect on his coronary artery disease or any role in triggering any of his heart attacks. Employer did not explicitly contend that the presumption should not be invoked with respect to the heart attacks, but it did insist that it rebutted the presumption the Claimant's heart attacks were related to his employment. Employer noted further that Claimant elected to discontinue the Plavix with knowledge of the risks in order to have the surgery and epidurals. Claimant's choice, Employer argued, should relieve it of responsibility for the heart attacks triggered by the consequences of his voluntary decision.

Beyond that, Employer asserted that even if it failed to rebut the presumption, the record establishes that the cardiac care Claimant is receiving now, and will need in the future, is the same care he would need absent anything that happened at work; and, therefore, it is not responsible for his future cardiac care. Emp. Br. at 10-15.

The Board has determined that, under circumstances in which a claimant's work plays any role in the manifestation of a disease, the entire resulting disability is compensable. *See*, Obert v. John T. Clark and Son of Maryland, 23 BRBS 157 (1990). Indeed, Employer acknowledged at the hearing that if any of Claimant's heart attacks related to treatment for his back condition, he has "a legitimate claim." Tr. 213-4. Further, if there is more than one causation issue, employer must address all possible elements. Zeigler v. Dept. of the Army/NAF, (BRB No. 99-0122) (Oct. 7, 1999) (Unpublished). In this instance, Claimant alleged several causation issues, and each will be addressed below, seriatim.

Sedentary Lifestyle

Dr. Joshua Furman, a specialist in internist medicine and cardiovascular disease, reviewed Claimant's medical records and evaluated his heart condition in a report dated January 19, 2006. He noted that Claimant suffered an MI on September 12, 1997, and underwent heart surgery on January 28, 1998. Thereafter, Claimant engaged in a vigorous exercise program at home and at work. Dr. Furman continued: "the impact of dynamic aerobic exercise on the cardiovascular system is well documented in terms of its protective effect, myocardial conditioning against ischemic events, reduction in basic blood pressure and heart rate, as well as impact on cardiovascular physiology including but not limited to endothelial function, vasodilation and myocardial oxygen consumption." The back injury on June 5, 1999, caused a decline in Claimant's ability to exercise, leading to a sedentary state, and, in Dr. Furman's opinion, eliminated the advantages he received from the exercise and increased his risk of a cardiovascular event. In Dr. Furman's opinion, the back injury triggered a "lack of activity" which "significantly contributed to both cardiac events in 2004." Dr. Neifeld, a specialist in internal medicine, agreed. He prepared a report dated January 17, 2006, in which he opined that a sedentary lifestyle caused by a back injury could cause or contribute to a worsening of a cardiac condition. These medical opinions linking Claimant's heart attacks to the injury-induced sedentary lifestyle are sufficient to invoke the presumption.

Celebrex

As a result of the spinal injuries, Claimant received anti-inflammatory medications, including Celebrex, from Drs. Castellvi and Dennison. Dr. Neifeld noted that FDA labeling specifically warns of the risk of a cardiac event with long term use of Celebrex, and, in his opinion, long-term use of Celebrex "probably" contributed to the cardiac events in 2004. Dr. Neifeld's opinion based upon the FDA warning is sufficient to invoke the presumption that Claimant's use of Celebrex contributed to his heart attacks in 2004.

Plavix

The discontinuation of Plavix involves a different issue. Unlike the sedentary lifestyle imposed by the spinal injury and the Celebrex which was prescribed as a treatment for the injury, Plavix was prescribed as a treatment for Claimant's underlying, pre-existing coronary artery disease. Further, the

gallbladder surgery in August of 2004, which required the suspension of Plavix, was unrelated to his spinal injury. Accordingly, to the extent that the discontinuation of Plavix for that surgery contributed to the heart attack in August of 2004, it was not related to the spinal injury.

Conversely, the epidurals in December, 2004, were administered as a treatment for the spinal injury. The record shows that as a prerequisite to the epidural treatments in December, 2004, Dr. Dennison recommended suspension of the Plavix. Dr. Gandhi, Claimant's cardiologist, warned against that, due to the increased risk of myocardial infarction related to discontinuing Plavix; however, after fully informing Claimant of the risks, he eventually authorized Claimant to proceed. Shortly after he discontinued the Plavix, Claimant, as Dr. Gandhi had warned, suffered a heart attack.

In Dr. Furman's opinion, the coronary events following both the gallbladder operation in August, 2004 and the epidurals in December, 2004, were "significantly affected" by the discontinuation of Plavix. He noted further that Plavix has not been discontinued since the December, 2004, thrombic event, and Claimant has not had another event. Dr. Neifeld agreed, noting in particular that the discontinuation of Plavix before the epidurals could have caused or contributed to a subsequent myocardial infarction. The record shows further that the staff at Tampa Hospital, where Claimant was treated for the December 26, 2004, heart attack, specifically attributed that event to the suspension of Plavix. Thereafter, Dr. Gandhi informed Claimant that he would not again provide clearance to stop the Plavix, and, subsequently, he declined a request that he do so.

Because it was necessary to discontinue the Plavix in order to permit the injury-related epidurals, the consequences must be deemed to have arisen necessarily out of the injury. Wheeler v. Interocean Stevedoring, Inc., 21 BRBS 33 (1988); *see also* White v. Peterson Boatbuilding Co., 29 BRBS 1 (1995); Mattera v. M/V Mary Antoinette, Pacific King, Inc., 20 BRBS 43 (1987); Weber v. Seattle Crescent Container Corp., 19 BRBS 146 (1986). The evidence is thus more than sufficient to invoke the presumption that the heart attack Claimant suffered on December 26, 2004, was induced by the suspension of Plavix under circumstances which render it a work-related incident associated with a prescribed medical treatment for that injury.

Rebuttal of Lifestyle Presumption

Dr. Sullebarger considered the contention that Claimant's sedentary lifestyle contributed to his MIs. He explained that the level of physical activity does not impact whether a patient gets blockages, and, in his opinion, Claimant's sedentary lifestyle "is not a major factor in the generation of his atherosclerosis," or its progression. Dep. at 37. He agreed that exercise benefits the coronary system and the way a patient feels, but, he noted, it does not he effect the blockages themselves. In his opinion, when a patient has an MI, he typically has a blockage someplace because it is unusual for a patient to have a cardiac event in the absence of a blockage. Further, the catheterizations after both MIs in 2004 showed the presence of such blockages.

Under these circumstances, it appears that Dr. Sullebarger has ruled out the injury-induced sedentary lifestyle as a cause of the blockages that triggered the cardiac events in 2004, and has, accordingly, rebutted the presumption not only under Brown v. Jacksonville Shipyard, but in accordance with rebuttal criteria applied in other circuits. *See*, Ortco Contractors, Inc. v. Charpentier, 332 F.3d 283, (5th Cir. 2003), cert. denied, 124 S. Ct. 825 (2003); Bath Iron Works Corp. v. Director, 137 F.3d 673, (1st Cir. 1998). Considering the evidence reviewed in its entirety, Claimant has failed to establish that his injury-related sedentary lifestyle caused or contributed to his 2004 MIs or his current coronary condition.

Rebuttal of Celebrex Presumption

Dr. Sullebarger addressed the contention that Celebrex contributed to Claimant's heart attacks. He observed that Celebrex has been shown to activate platelets and increase the risk of a thrombic event. He noted further, however, that with Celebrex it is unusual for a patient to have an event if there is no blockage, and typically when an event occurs, the patient has a blockage someplace. Dr. Sullebarger observed that many factors contributed to Claimant's heart attacks and noted that the events happened on the days they did "because he had these procedures being done;" but he was unsure of the "relative contribution of all those things," and acknowledged that Celebrex may have tipped the balance.

Under circumstances in which Claimant has invoked a presumption that a drug prescribed to him for a work-related injury contributed to heart attacks he suffered in August of 2004, and December of 2004, Dr. Sullebarger's candid acknowledgment that he is unsure of the drug's relative contribution to the cardiac incidents, and his observation that it may have tipped the balance, is plainly

insufficient to rebut the presumption of a causal link. Although Claimant's underlying coronary artery disease may be the primary cause of his heart attacks, the Board has held that the presumption is not rebutted under circumstances in which employer failed to rule out a second, work-related cause which contributed to or accelerated the disease process. Peterson v. General Dynamics Corp., 25 BRBS 71 (1991), aff'd sub nom. Ins. Co. of N. America v. U.S. Dept. of Labor, 969 F.2d 1400, (2d Cir. 1992), cert. denied, U.S., 113 S.Ct. 1253 (1993)(smoking primary cause of cancer; asbestos exposure secondary, contributing factor).

Since Celebrex was a medication prescribed for the 1999 job-related injury which has not been ruled out as a factor contributing to Claimant's August, 2004 heart attack, I find that Employer has failed to rebut the Section 20(a) presumption under Brown v. Jacksonville Shipyard. Further, Dr. Sullebarger, rather than suggesting that Celebrex was not a causal factor, seemed more inclined toward the position that Celebrex may have tipped the causal balance and contributed to the MIs. As such, the presumption also has not been rebutted under a substantial evidence review considering the record viewed in its entirety. *See*, Ortco Contractors, Inc. v. Charpentier, 332 F.3d 283, (5th Cir. 2003), cert. denied, 124 S. Ct. 825 (2003); Bath Iron Works Corp. v. Director, 137 F.3d 673, (1st Cir. 1998).

Rebuttal of the Plavix Presumption

The record shows that Claimant was taken off his antiplatlet drug, Plavix, from time to time, and, each time he ran the risk of triggering a thrombic event. Dr. Sullebarger observed, however, that not everyone who stops taking Plavix has a heart attack, and he noted that Claimant had stopped Plavix several times without incident. He explained further, however, that it is unusual for a patient to have an event if there is no blockage, but he acknowledged that, like taking Celebrex, the suspension of Plavix may have tipped the balance. Taking into consideration Claimant's cardiac history, Dr. Sullebarger forthrightly noted that he could not fairly concluded that the cardiac events in 2004 were the inevitable progression of the coronary artery disease, and he concluded that one reason, among others, that contributed to the MIs was: "because he had these procedures being done."

Since the epidural procedures in December of 2004, were treatments directly related to the 1999 spinal injury, Dr. Sullebarger's observation that the thrombic event was triggered either by the suspension of Plavix to permit these epidurals or because he had these epidurals establishes a work-related etiology for the December 26, 2004 heart attack rather than ruling out an injury-related causal link. Since the suspension of Plavix to facilitate treatment of a work-related injury has

not been ruled out as a factor contributing to Claimant's December 26, 2004 heart attack, I find that Employer has failed to rebut the Section 20(a) presumption under Brown v. Jacksonville Shipyard. Further, Dr. Sullebarger, rather than suggesting that the suspension of Plavix was not a causal factor, seemed more inclined toward the position that stopping Plavix may have tipped the causal balance and contributed to the MIs. As such, the presumption also has not been rebutted under a substantial evidence review considering the record viewed in its entirety. *See*, Ortco Contractors, Inc. v. Charpentier, 332 F.3d 283, (5th Cir. 2003), cert. denied, 124 S. Ct. 825 (2003); Bath Iron Works Corp. v. Director, 137 F.3d 673, (1st Cir. 1998).

Voluntary Suspension of Plavix

Employer argues further, however, that it should not held responsible for heart attacks caused by Claimant's voluntary decision to discontinue Plavix when his own cardiologist warned him that by stopping it, he substantially elevated the risks of triggering a cardiac event. Employer thus suggests that Claimant accepted the risk and the responsibility for any consequences of suspending his Plavix when he agreed to undergo the epidural injections recommended by Dr. Dennison. Leaving aside the presumed contribution of Celebrex to Claimant's 2004 MIs, the case law does not otherwise support Employer's contention.

The circumstances here are clearly distinguishable from the line of cases which hold that a claimant's misconduct, intentionally harmful conduct, or carelessness in regard to his injury may constitute a non-work-related intervening event. *See*, Bludworth Shipyard Inc. v. Lira, 700 F.2d 1046 (5th Cir. 1983); Cyr v. Crescent Wharf and Warehouse Co., 211 F.2d 454 (9th Cir. 1954); Grumbley v. Eastern Associated Terminals Co., 9 BRBS 650 (1979); Wright v. Connolly-Pacific Co., 25 BRBS 161, 164 (1991), aff'd mem. sub. nom. Wright v. Director, 8 F.3d 34 (9th Cir. 1993); Konno v. Young Brothers, Ltd, 28 BRBS 57, 63 (1994). Indeed, this record is devoid of any evidence suggesting that Claimant intended to harm himself when he elected to discontinue the use of Plavix. True, he understood the risk as explained to him by Dr. Gandhi, but the medical procedure that necessitated the suspension of Plavix was recommended by Dr. Dennison to treat his injury; and his cardiologist authorized him to discontinue the Plavix to facilitate the treatment prescribed by Dr. Dennison.

Under these circumstances, Employer has clearly failed to establish that Claimant intended to harm himself or acted carelessly when he decided to discontinue the Plavix. Claimant did what he could to inform himself of the risks.

He consulted with his cardiologist who authorized him to proceed, and he was following the advice of his pain care physician; thus refuting any suggestion that Claimant acted in reckless disregard of his own health and safety. *See,* Jones v. Director, 977 F.2d 1106 (7th Cir. 1992); James v. Pate Stevedoring Co., 22 BRBS 271 (1989). In this instance, the record shows that Claimant acted reasonably with due regard for his injury and his heart condition.

Indeed, even in a situation involving a failure to follow prescribed medical treatment, the Board has held that the presumption is not rebutted. Ogundele v. American Sec. & Trust Bank, 15 BRBS 96 (1980). Logically, then, if a refusal to follow medical advice is insufficient to establish a willful intent to injure oneself, Jackson v. Strachan Shipping Co., 32 BRBS 71 (1998), it would seem to follow that a claimant who accepts a risk associated with an injury-related recommended medical procedure does not relieve employer of responsibility if the risk becomes a reality.

Thus, the Board has held that a physician's treatment of a claimant's work injury, even if it was unnecessary and constituted malpractice, does not sever the causal relationship between the injury and the employment. Wheeler v. Interocean Stevedoring, Inc., 21 BRBS 33 (1988); *See*, Caudill v. Sea Tac Alaska Shipbuilding, *supra.* To the contrary, if claimant's choice of physician and treatment are reasonable, claimant may receive benefits for any increased disability due to the treatment provided. Wheeler, *supra*; *see also*, White v. Peterson Boatbuilding Co., 29 BRBS 1 (1995); Mattera v. M/V Mary Antoinette, Pacific King, Inc., 20 BRBS 43 (1987)(back sustained while undergoing vocational testing in connection with his work-related arm injury necessarily arose out of and in the course of employment); Weber v. Seattle Crescent Container Corp., 19 BRBS 146 (1986)(neck injury sustained during the course of medical examination scheduled at employer's request for work-related hearing loss claim is covered under the Act). Lindsay v. George Wash. Univ., 279 F.2d 819 (D.C. Cir. 1960); see also Austin v. Johns-Manville Sales Corp., 508 F. Supp. 313 (D. Me. 1981). Consequently, Employer is not relieved of responsibility for the December 26, 2004 heart attacked merely because Claimant, knowing the risks, decided to discontinue his Plavix to permit the epidural injections for his back injury.

Heart Damage

The record shows that Claimant was admitted to Brandon Regional Hospital in early August, 2004, for a heart attack following gallbladder surgery. The hospital discharge summary dated August 9, 2004, reveals that a catheterization

showed, *inter alia*, total occlusion of the right coronary artery and of the vein graft to the first marginal "which was successfully recanalized…." Later that year, Claimant was admitted to Tampa General Hospital after suffering a heart attack on December 26, 2004. Dr. Cintron reported in the hospital discharge summary that a catheterization on December 29, 2004, noted a clot in the circumflex artery in the mid-distol location which was treated with angioplasty and stenting. Hospital records indicated that the mid-circumflex artery clot was "most likely secondary to the patient stopping Plavix." Cx 12 at 489; CX 20.

Dr. Sullebarger reviewed the echocardiogram and EKG data. He noted that a pre-operative stress test before the gallbladder surgery in July, 2004, showed an ejection fraction, which is a measure of the heart's ability to pump, of 67%. An ejection test in August, following the first of the two 2004 heart attacks, was 60%. After the December 26, 2004 heart attack, Claimant's ejection fraction went down to 58%. Dr. Sullebarger noted that while the difference between an ejection fraction or 60 and 58 is probably within the margin of error for the test, a reduction from 67% to 60% or 58% indicates a definite decline. Still, he characterized an ejection fraction of 58% as slightly below normal which is 60-plus. In his opinion, the ejection fraction of 58% shows a small problem or minor damage, and is actually above Claimant's 1998 ejection fraction which was in the low 40's.

Dr. Sullebarger explained further, however, that each heart attack causes some permanent cell damage which is never replaced, and each is significant. He agreed that the MIs in 2004 may have resulted in damage, and if they did, the damage was permanent, but "slight," and their impact on Claimant's lifestyle was "trivial;" however, he was unable to determine whether the damage seen in 2006 was the result of the 1998 incident or the subsequent smaller heart attacks.

Medical Benefits for Coronary Condition

Hospitalization and Temporary Adjustments in Baseline Care

As discussed above, the heart attacks Claimant suffered on August 9, 2004, and December 26, 2004, were work-related events, and, as such, he is entitled to the medical benefits the Longshore Act provides for the care and treatment of covered injuries. In this instance, these include the August 9, 2004, admission to the Branden Regional Hospital, the catheterization and other care and treatment provided by that facility during his hospitalization. Indeed, Dr. Sullebarger agreed that the catheterization following the August 9, 2004, cardiac incident showed a

total right occlusion that did not previously appear, and that it required treatment different from the treatment Claimant had received before. The record further shows that the visits with Dr. Gandhi on August 4, 2004, and September 29, 2004, and to the Brandon Cardiology Clinic on November 2, 2004, were, as their office notes reveal, follow-up visits during which Claimant's progress following his August 9, 2004 heart attack was monitored by his physicians. Beyond that, however, the record does not indicate that Claimant received any other temporary care or treatment associated with that event, and he seems to have reverted to his baseline treatment.

On December 26, 2004, Claimant suffered another heart attack. As discussed above, this event, too, was triggered by the work related injury, and Claimant is entitled to the medical benefits for his admission to Tampa General Hospital, the catheterization and other care and treatment provided by that facility during his hospitalization. Again, Dr. Sullebarger agreed that the catheterization following the December 26, 2004, cardiac event showed a clot in the circumflex artery that was treated with angioplasty and stenting. The record further shows that the visits with Dr. Gandhi on January 5, 2005, April 18, 2005, at which time Claimant stopped taking Norvasc, and April 27, 2005, when Dr. Gandhi stopped the diuretics and reduced the beta-blocker back to 50 mg. All of these visits, as Dr. Gandhi's office notes reveal and Dr. Sullebarger acknowledged, were follow-up visits during which Claimant's progress following his December 26, 2004, heart attack was monitored. After April 27, 2005, however, the medical record does not indicate that Claimant received any other temporary care or treatment associated with that event, and he seems to have reverted again to his baseline treatment.

The record shows that the myocardial events in 2004 contributed to Claimant's need for cardiac care, temporarily, his hospitalizations, and the temporary follow-up care and medications. Further since these MIs, as discussed above, are work-related, the Claimant is entitled to medical benefits under the Longshore Act for the temporary care and treatment he received for these events.

Long Term Baseline Care

Dr. Sullebarger also emphasized, however, that Claimant's long term care and treatment remained the same before and after the 2004 MIs. Based on his review of the record, Dr. Sullebarger concluded that Claimant's medications were only adjusted temporarily following the 2004 cardiac incidents. He noted, for example, that Claimant has needed treatment since 1998 for blood pressure, cholesterol, blood sugar, and atherosclerotic disease, and that his need continues.

According to Dr. Sullebarger, now that Claimant has recovered from his two MIs, he reverted to the same care and medications would need had the 1999 back injury not occurred and Claimant had undergone none of the procedures and took none of the medications associated with that injury. As he describes it, what Claimant needs now for his atherosclerosis was set in motion in 1997.

Claimant, of course, testified that some of his medication doses increased and some new medications were added, and argued that Dr. Sullebarger only had Dr. Gandhi's records through April of 2005; however, Claimant's treating cardiologist has not addressed this issue, at least not in this record, and has provided no comparison of the long-term treatment alterations he prescribed, if any, following the two MIs in 2004. Moreover, the November 2, 2004 report by the Brandon Clinic noted over twenty medications Claimant takes in various doses, and other than the Norvasc, the beta-blocker, and the diuretic mentioned above, neither Claimant in his testimony, nor the record as a whole, identifies any other specific long term change in his medications. As a result, the record is devoid of medical evidence which in any way refutes Dr. Sullebarger's assertion that any cell damage caused by the cardiac events in 2004 was minor, and resulted in only temporary changes in Claimant's pre-existing, baseline need for medication and his continuing long term care.

Nevertheless, Claimant argues that his cardiac care is now totally Employer's long-term responsibility. Citing case law he deems applicable, Claimant argues that Employer is responsible for all of his cardiac care, because an employment-related injury contributed to, combined with or aggravated a pre-existing condition, rendering the entire resultant disability compensable. Independent Stevedoring v. O'Leary, 357 F.2d 812 (9th cir. 1966); Rajotte v. General Dynamics, 18 BRBS 85 (1986); *see also*, Mijangos v. Avondale Shipyards, Inc., 19 BRBS 15 (1986), rev'd on other grounds, 948 F.2d 941, (5th Cir. 1991). Although medical benefits, not disability compensation, is the issue here; the situations are analogous.

While Claimant accurately cites the general rule, the particular facts involved here present a very close case; and I have found no case directly on point. The record shows that the two work-related MIs necessitated temporary alterations in Claimant's long term care and treatment, but the medical evidence also shows that he eventually returned to the baseline treatment he was receiving for his pre-existing condition before those two events occurred. As a result, a case like Gooden v. Director, 135 F.3d 1066 (5th Cir. 1998), is similar but distinguishable. In Goodin, for example, the Court noted the claimant's work may not have caused

or aggravated his cardiovascular disease, but a work-related heart attack is compensable, and concluded that it is erroneous to focus on the origins of the underlying condition rather than on the ultimate heart attack. In a slightly different context, the Court in Atlantic Marine v. Bruce, 661 F.2d 898, (5th Cir. 1981), aff'g 12 BRBS 65 (1980) found an employer liable for all medical expenses for a myocardial infarction which were the natural and unavoidable result of the work injury, and not due to an intervening cause. In this instance, it seems that the long term medication and treatment are not the natural and unavoidable result of the work injury, but are prescribed for Claimant's pre-existing coronary artery disease.

Yet, Dr. Sullebarger explained that each heart attack causes some permanent cell damage which is never replaced, and each is significant. He agreed that the MIs in 2004 may have resulted in damage, and if they did, the damage was permanent, but "slight," and their impact on Claimant's lifestyle was "trivial." He did note, however, that Claimant's ejection fraction prior to the first 2004 MI was 67% and it fell to 60% after the August, 2004, MI, and to 58% following the December, 2004 MI. He further described the reduction from 67% to 58% as definitely indicative of a worsening in Claimant's condition. Still, Dr. Sullebarger explained that the ejection fraction of 58% as slightly below normal which is 60-plus. In his opinion, the ejection fraction of 58% shows a small problem or minor damage, and it actually is above Claimant's 1998 ejection fraction which was in the low 40's. Dr. Sullebarger, however, was unable to determine whether the damage seen in 2006 was the result of the 1998 incident or the subsequent smaller heart attacks.

It is reasonable to conclude from Dr. Sullebarger's analysis that Claimant had exhibited improvement after 1998, then suffered some indeterminate heart damage as a consequence of the two MI's in 2004, which, based upon ejection fraction data, set him back and worsened his cardiac condition, just not to the level it was in 1998. Dr. Sullebarger testified further that each cardiac event contributes to the need for cardiac care. Again the parties have cited no case law directly on point, but the most closely analogous principles seem to be articulated by the Board in Turner v. Potomac Telephone Co., 16 BRBS 255 (1984), which held that treatment is compensable even though it is due only partly for a work-related condition, and Kelley v. Bureau of National Affairs, 20 BRBS 169, 172 (1988), which held that where relevant evidence establishes that a claimant's condition was occasioned, at least in part, by his work injury, treatment received for the condition was compensable under the Longshore Act.

In this instance, it seems reasonable to infer that Claimant, who required a baseline level of coronary care before two work-related MIs, surely required at

least that much care following those two heart attacks which reduced his ejection fraction from 67% to 58%; and there is no dispute among the medical experts that the two MIs contributed to his need for cardiac care. Under such circumstances, Kelly and Turner suggest that all of the cardiac care and treatment Claimant receives is compensable under the Longshore Act. Therefore:

ORDER

IT IS ORDERED that the claim for medical benefits for Claimant's pulmonary condition, designated as 2003 LHC 1686, (OWCP # 06-179845), be, and it hereby is, denied, and;

IT IS FURTHER ORDERED that the claim for medical benefits for Claimant's cardiac condition, designated as 2005 LHC 2089, (OWCP # 06-187024), be and it hereby is, granted.

A

Stuart A. Levin
Administrative Law Judge